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ABSTRACT

This experiment was designed to discriminate among two diametrically opposed states, arousal and relaxation, which have been attributed to alcohol ingestion. Male social drinker subjects were assigned to form two independent groups of ten subjects each. Baseline measure of heart rate, skin conductance level (SCL), pulse wave amplitude and ear lobe temperature were recorded. Group I then received .3 ml/lb of body weight pure ethanol in orange juice over a five-minute period. Group II received 1 ml of ethanol floated on top of juice, and both groups' physiological measures were continuously recorded over the next 40 minutes. Results showed that reliable effects were not obtained with the SCL measure, and that heart rate increased reliably but nondifferentially for both groups. Ear lob temperature increased for the alcohol and decreased for the placebo group. It was concluded that, in the dose used, alcohol acted as a relaxant, and that previous experiments found equivocal and conflicting results because they did not utilize a placebo control group but drew inferences from within subject baseline to post-drink comparisons. (Author/PC)

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PHYSIOLOGICAL CONCOMITANTS OF THE ALCOHOL STATE: AROUSAL OR RELAXATION

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ABSTRACT: *The present experiment was designed to discriminate among two diametrically opposed states, arousal and relaxation, which have been attributed to alcohol ingestion. Male social drinker subjects matched on age, drinking history, socioeconomic level and at least four hours food and stimulant deprived were assigned to form two independent groups of ten subjects each. Baseline measures of heart rate, skin conductance level, pulse wave amplitude and ear lobe temperature were recorded. Group I then received 0.3 ml/lb of body weight pure ethanol in 4:1 orange juice mix over a paced five minute period. Group II received an equivalent amount of orange juice per unit of body weight with 1 ml of ethanol floated on top. Physiological measures were continuously recorded over the next forty minutes. Groups did not differ on any of the measures during baseline suggesting that adequate matching on the stated parameters was obtained. Reliable effects were not obtained with the SCL measure. Heart rate increased reliably but nondifferentially for both groups. Ear lobe temperature increased for the alcohol and decreased for the placebo group. Initially, zero minutes post drink ingestion pulse wave amplitude decreased reliably and nondifferentially for both groups with respect to baseline. Five minutes post drink the alcohol group demonstrated a strong vasodilation effect which continued for thirty-five minutes. The placebo group stayed constricted and never reached baseline. It was concluded that in the dose used alcohol is a relaxant and that previous experiments found equivocal and conflicting results because they did not utilize a placebo control group, but drew inferences from within subject baseline to post drink comparisons.*

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Ludwig (1966, 1971) has comprehensively reviewed the techniques of inducing altered states of consciousness. He points out that drugs are one of the most reliable ways of manipulating state. James (1882) and Ritchie (1965) have observed that alcohol is one of the oldest and most often used drugs to manipulate state.

Psychophysiological techniques have been used for a number of years to index and evaluate a variety of states such as sleep stages, activiation and arousal levels, fear, anger and the physiological effects of alcohol ingestion (Naitoh, 1972). There is good agreement among experimenters that alcohol ingestion induces an altered state as indexed by a variety of psychophysiological measures. However, there is less agreement among experimenters as to the exact nature of the relationship. Two diametrically opposed premises have guided much of the research relating psychophysiological measures to alcohol ingestion.

The first premise holds that alcohol makes people feel relaxed and less tense (Carpenter, 1957; Greenberg & Carpenter, 1957; McGonnell & Beach, 1968; Kissen, Schenker & Schenker, 1959). It has been suggested that such alcohol induced tension reduction may be the motivation for the widespread use of alcohol. The second premise holds that alcohol ingestion leads to arousal and activation (Perman, 1958; Docter & Perkins, 1961; Morikawa, et.al., 1968; Walsh, 1971).

Earlier experiments have come up with equivocal and at times conflicting results, in part, because of methodological differences. Very few experiments have utilized placebo control groups, the same psychophysiological measures or the same dose of alcohol. Thus, both arousal and relaxation hypotheses are still being entertained. It was the purpose of the present experiment to discriminate between arousal and relaxation hypotheses of alcohol ingestion by utilizing a battery of physiological measures, a placebo control group and controlling for sex, age, drinking history, socioeconomic level and time since last ingestion of food and stimulants.

Method

Subjects. --The subjects were two independent groups of adult males matched on age, drinking history and socioeconomic status. Experimental and control groups did not differ reliably on any of the matching factors i.e., age 27.6 vs 26.0 years; income $6500 vs $5340; or alcohol consumption (the equivalent of three cans of beer or less/week for both groups).

Procedure. --Subjects were told not to drink alcoholic beverages on the night before the experiments, to get a good nights sleep and to skip stimulants and breakfast. Subjects came to the laboratory at least four hours food and drink deprived. After having the experiment explained to them subjects signed informed consent forms, were weighed and took a practice breathalyzer sample (to determine if in fact they had zero blood alcohol cencentrations at the start of the experiment). Subjects filled out a questionnaire and were then instrumented to record heart rate, skin conductance, ear lobe temperature and photoplethysmographic pulse wave amplitude on a Beckman dynograph. Baseline measures were taken for ten minutes. Experimental subjects then received 0.3 ml of pure ethanol in a 4:1 orange juice mix/lb of body weight. Control subjects received an equivalent dose of orange juice per unit of body weight with 1 ml of ethanol floated on top of the drink. The drink was ingested over a paced five minute period. Autonomic measures were continuously recorded over the next forty minutes. Alcoholized and control subjects received a breathalyzer test at the end of the forty minutes to determine peak blood alcohol concentrations attained.

Response quantification

Skin conductance level. --Baseline SCL was measured as the mean of five once per minute samples at the 6th, 7th, 8th, 9th, and 10th minutes of the baseline period. Post drink SCL measures are based on the mean of nine measurements coming at the 0, 5th, 10th, 15th, 20th, 25th, 30th, and 40th minutes post drink.

Heart rate. --Baseline heart rate was quantified as the average of five ten second periods coming after the 6th, 7th, 8th, 9th, and 10th minutes of baseline. Post drink heart rate was quantified as the mean of nine ten second periods coming after 0, 5, 10, 15, 20, 25, 30, 35, and 40 minutes post drink.

Heart rate variability. --Was defined as the range of heart rates in one minute periods during the 6th, 7th, 8th, 9th, and 10th minutes of baseline. Post drink heart rate variability was defined as the heart rate range in one minute segments 0, 5, 10, 15, 20, 25, 30, 35, and 40 minutes post drink.

Ear lobe temperature. --Ear lobe temperature was taken throughout the experiment but was valid only in the drink and post drink period because prior to that Ss wore ear phones. Post drink ear lobe temperature was quantified as instantaneous readings 0, 5, 10, 15, 20, 25, 30, 35, and 40 minutes post drink.

Photoplethysmographic pulse wave amplitude. --Baseline pulse wave amplitude (PWA) was recorded as the mean of five ten second samples 6, 7, 8, 9, and 10 minutes after start of baseline. Post drink PWA was quantified as the mean percent of baseline 0, 5, 10, 15, 20, 25, 30, 35, and 40 minutes post drink (10 second samples).

Results

Figure 1 summarizes the skin conductance results. A 2 X 2 factorial ANOVA was performed on the data. The between groups (experimental-placebo) factor was not significant. The trials (baseline-post drink) effect was marginally significant ($.05 < p < .10$). T Tests indicated that the placebo control group did not increase its SCL from baseline to post drink reliably. However, the experimental group showed a reliable baseline to post drink SCL increase ($t = 2.59$, $df = 9$, $p < .05$).

Figure 2 summarizes the heart rate results. A similar 2 X 2 factorial was performed on the heart rate data. The groups did not differ in average heart rate. Baseline to post drink heart rate increased reliably ($F = 47.75$, $df = 1,18$, $p < .001$). The groups x trials interaction was not reliable, suggesting that heart rate increased equally for both placebo and alcohol groups from baseline to post drink.

Heart rate variability did not differentiate between groups nor did it show any reliable changes over trials.

Figure 3 presents the photoplethysmographic pulse wave amplitude results as a percent of baseline. The results were analyzed by a 2 X 2 ANOVA. The groups effect was reliable ($F = 5.53$, $df = 1,18$, $p < .05$). The alcohol group was more dilated than the placebo control group. The trials effect (zero and 40 minutes post drink ingestion) was highly reliable ($F = 25.90$, $df = 1, 18$, $p < .001$) indicating that dilation occurred overall. A significant groups by trials interaction was demonstrated ($F = 9.38$, $df = 1, 18$, $p < .01$). Figure 4 elucidates the nature of the relationship more clearly. Zero minutes post drink both alcohol and placebo groups showed a dramatic constriction such that response amplitude was only 50% of baseline. At this point the groups did not differ from each other. Five minutes post drink the alcohol group demonstrated a strong vasodilation effect while the placebo controls had not changed much at all. Thirty-five minutes later the alcohol group was still dilating while the control group showed a slight return toward baseline; a change probably due to relaxation in the experiment.

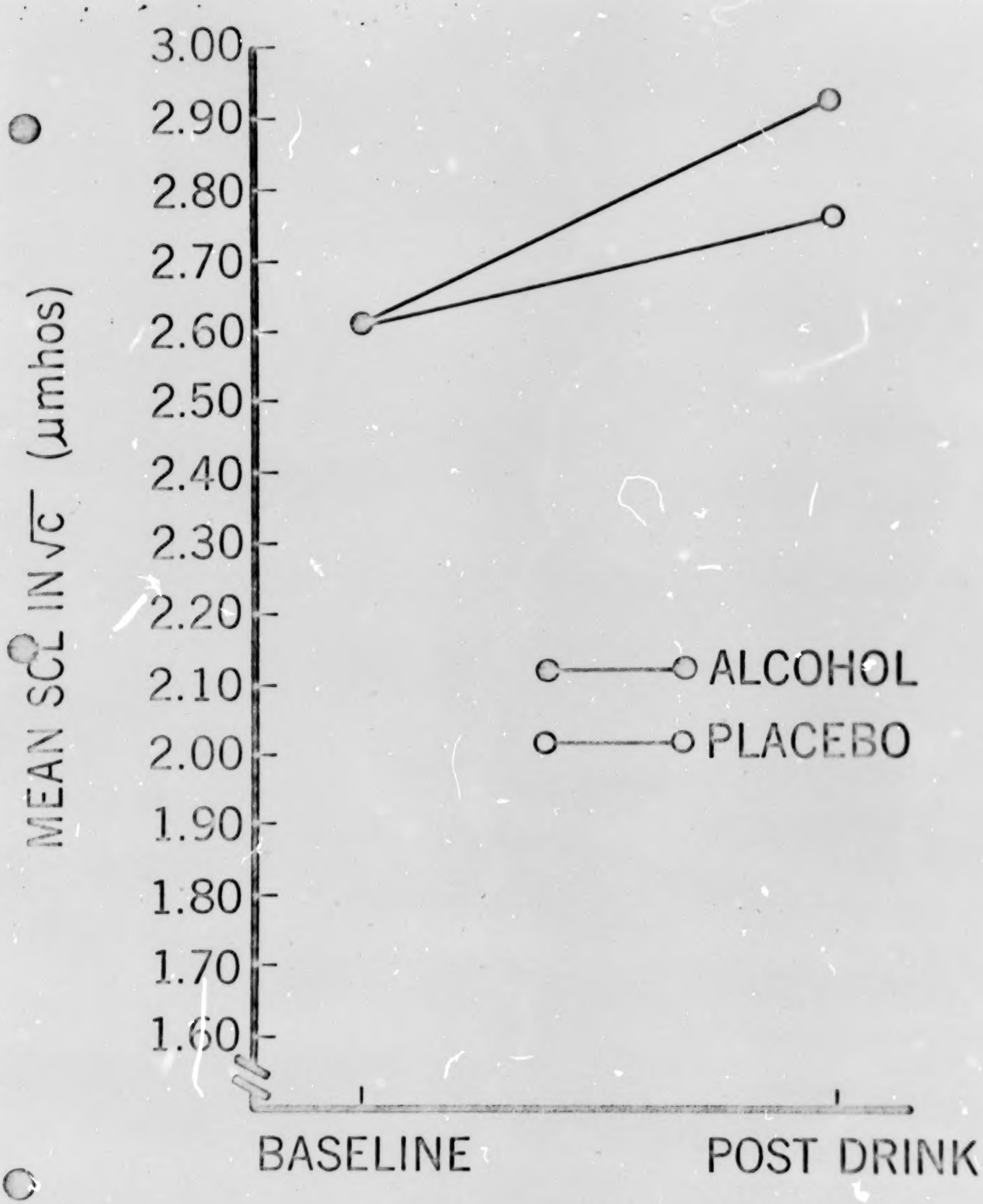

Figure 1. Mean skin conductance level at baseline and post drink conditions for alcohol and placebo groups (N = 10 each).

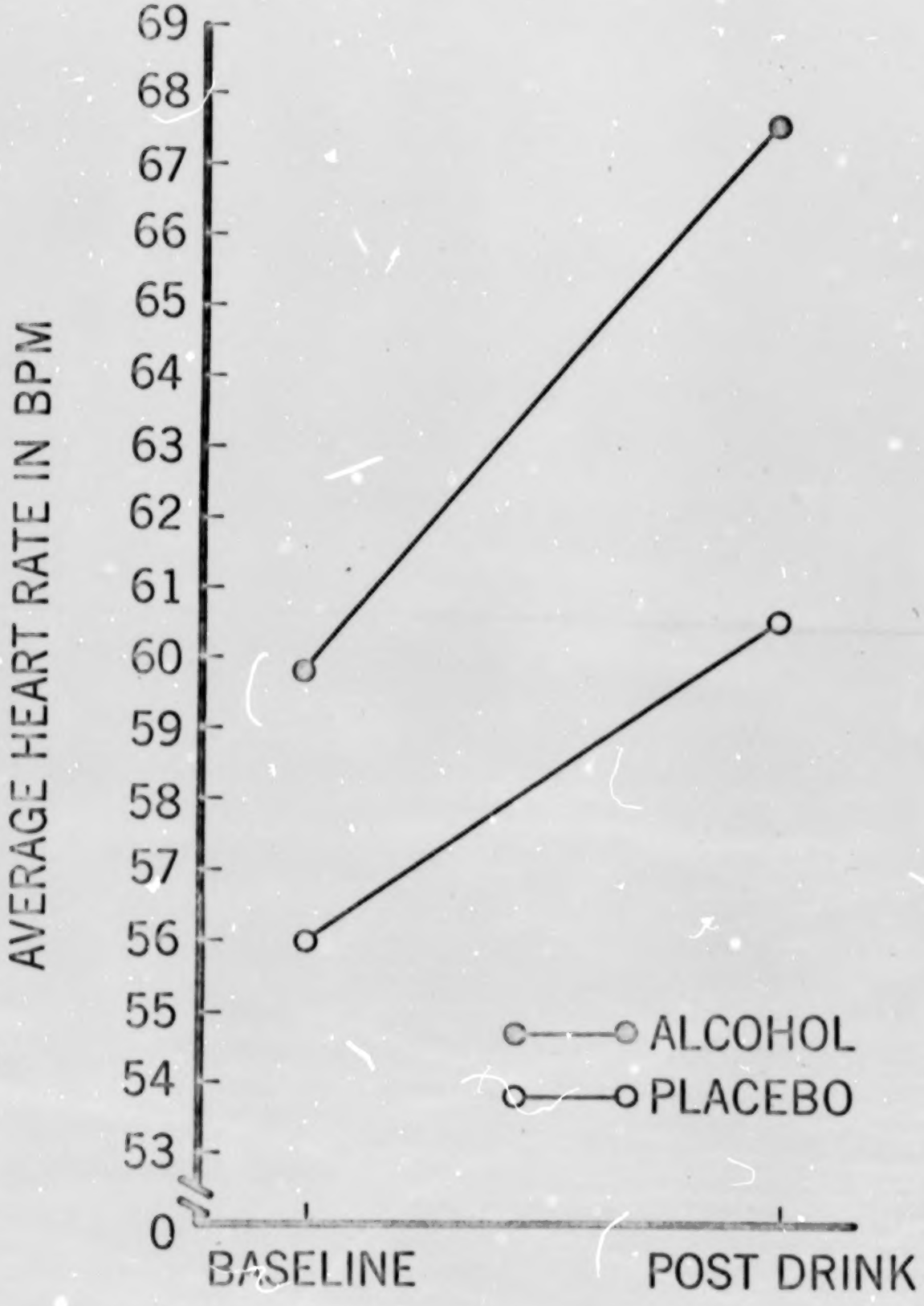

Figure 2. Mean heart rate levels over baseline and post drink conditions for alcohol and placebo groups (N = 10 each).

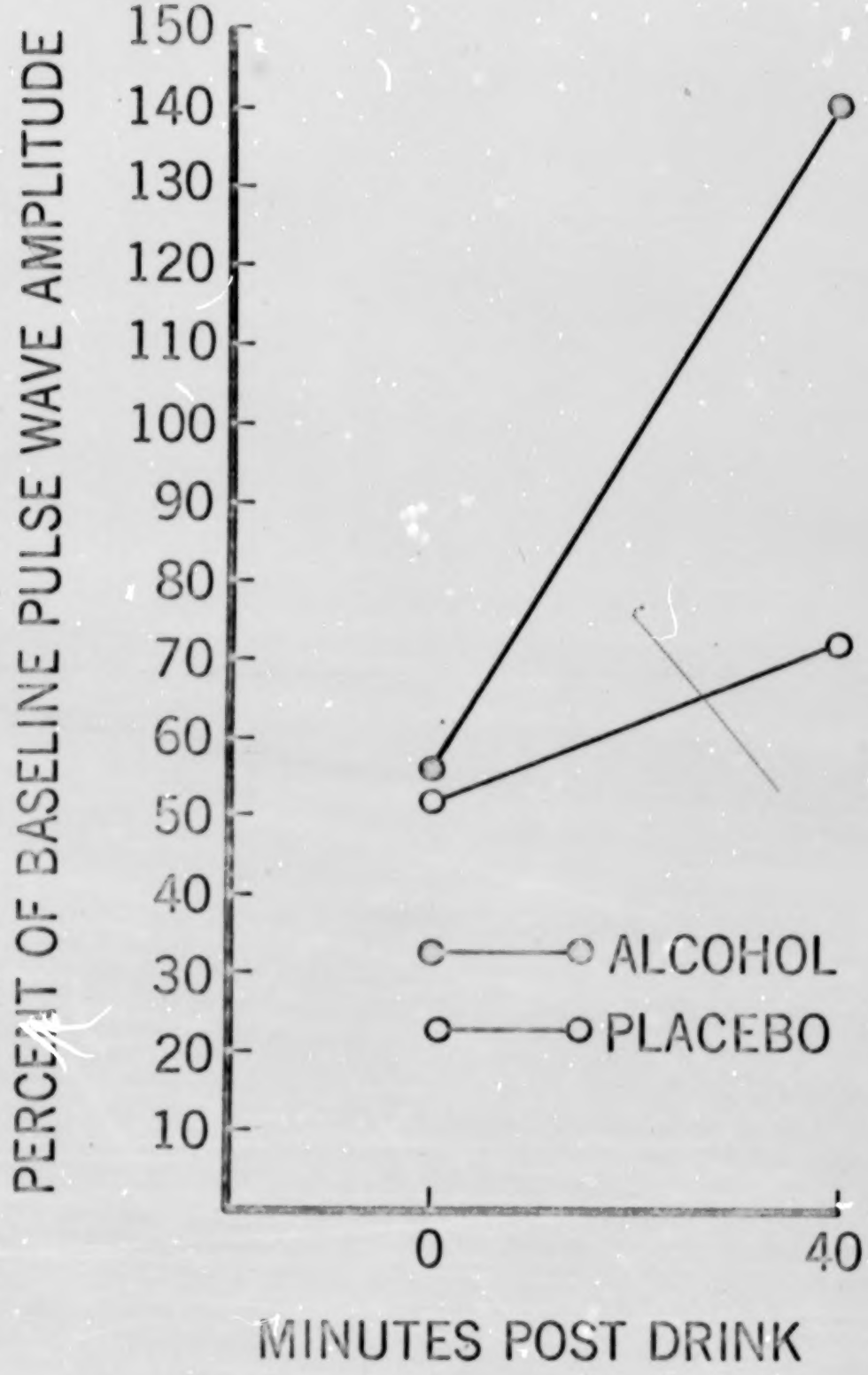

Figure 3. Percent of baseline pulse wave amplitude at 0 and 40 minutes post drink for alcohol and placebo groups (N = 10 each).

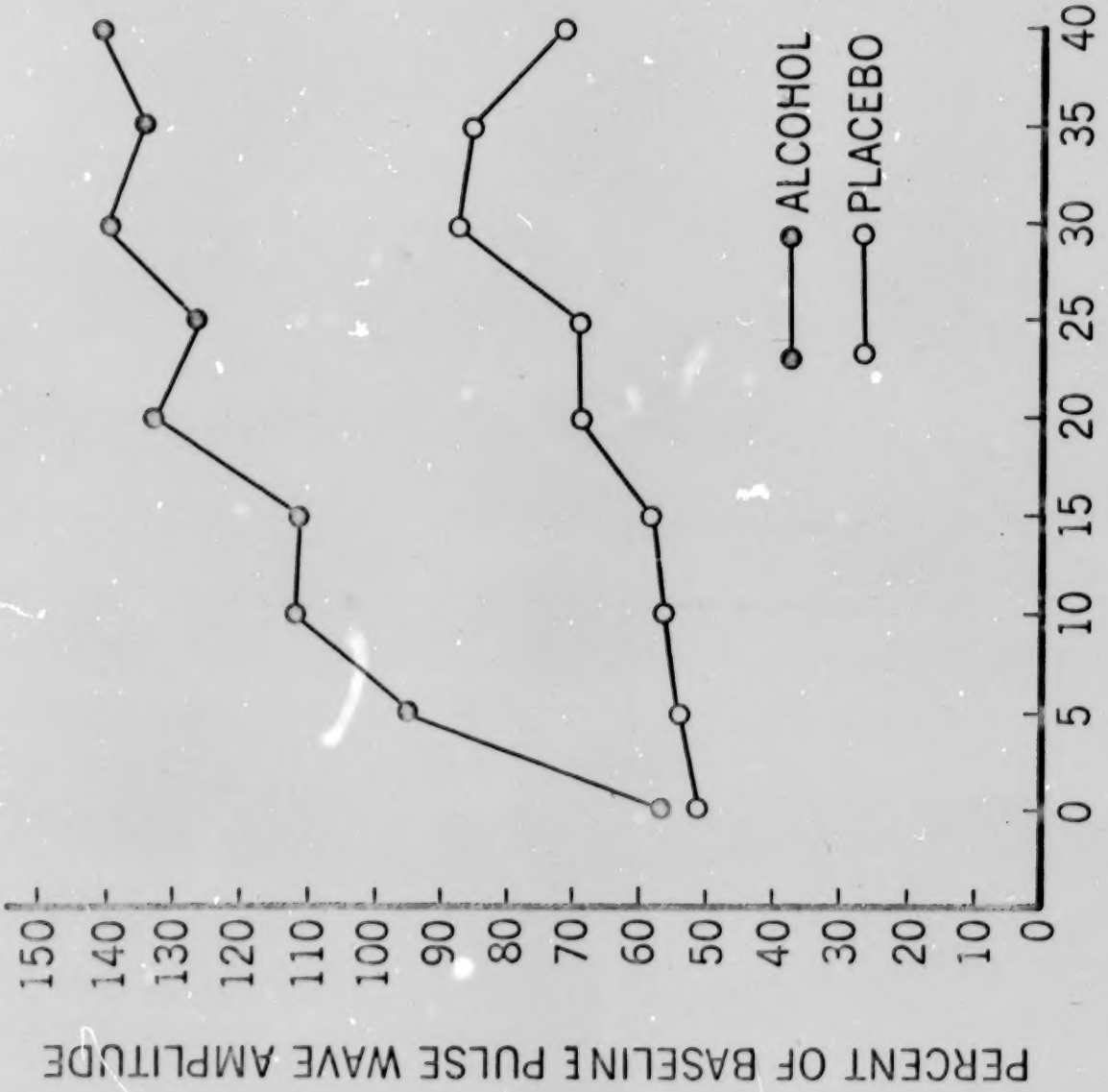

Figure 4. Percent of baseline pulse wave amplitude over five minute post drink blocks for alcohol and placebo groups (N = 10 each).

Ear lobe temperature data were analyzed by a 2 X 2 factorial analysis of variance. There was a significant groups effect ($F = 5.03$, $df = 1,18$, $p < .05$). No trial effect was demonstrated but the groups by trials interaction reached marginal levels of significance ($F = 3.85$, $df = 1,18$, $< .05$ $p < .10$). Further tests indicated that the groups did not differ reliably in ear lobe temperature zero minutes post drink ingestion. However, forty minutes post drink ingestion the alcohol group had a reliably higher ear lobe temperature than did the placebo control group ($t = 2.47$, $df = 1,8$, $p < .05$). The reliable difference forty minutes post ingestion came about because the alcohol group increased slightly and the control group decreased slightly, yielding in combination the reliable difference.

Discussion

Results of the present experiment support earlier experiments in indicating that there are changes in autonomic activity symptomatic of an altered state after ingestion of alcohol when compared with baseline measures. The present experiment differs from prior experiments because of the addition of a placebo control group (subjects received the orange juice carrier medium plus 1 cc of ethanol floated on top). A baseline versus post drink comparison would lead to the conclusion that ethanol increases tonic skin conductance levels. However, an experimental versus control group comparison does not support such an interpretation. Both groups recorded slight SCL increases which were neither differential nor reliable. Similarly, with the heart rate data, a comparison of baseline versus post drink heart rates would lead to the conclusion that alcohol has activating effects on heart rate. However, again such a conclusion is not supported by an alcohol versus placebo group comparison. Both alcohol and placebo groups showed reliable heart rate increases but there was no interaction to signify a differential effect. This finding suggests that the increased heart rates observed in both groups may have been due to cold orange juice in an empty stomach rather than a specific effect of alcohol. Such an interpretation of the heart rate data is in line with results reported by Docter and Perkins (1961) who also used a placebo control group and did not demonstrate differences between placebo and alcohol groups.

Since there were no significant group differences with heart rate and skin conductance measures, but pulse wave amplitude and temperature increased in the alcohol group while declining in the placebo control group, the results suggest that the specific effect of ethanol in the dose used is that of a relaxant. This is in line with verbal reports of the subjects suggesting feelings of warmth, drowsiness and relaxation. Further experiments are needed utilizing a dose-response analysis to test whether ethanol uniformly works as a relaxant or whether there are dose dependent arousing and or relaxing effects.

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